TONSILLAR ABSCESS

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This article presents seven cases of patients with tonsillar abscess formation and discusses the pathophysiology of intratonsillar abscess formation. (*J Natl Med Assoc.* 1991;83:333-336.)

Key words • peritonsillar abscess • intratonsillar abscess • tonsillitis • pharingitis

Identification of a tonsillar abscess following tonsillectomy au chaud for a peritonsillar abscess prompted a review of tonsillar abscesses. References to tonsillar abscess are scanty.[1,2] A review of peritonsillar abscess patients treated with tonsillectomy au chaud was undertaken to further delineate a tonsillar abscess and its relationship to peritonsillar abscess.

PATIENTS AND METHODS

A retrospective study was conducted of 148 patients with peritonsillar abscess between 1982 and 1987. Eighty-nine patients were hospitalized, and the remaining 59 were treated as outpatients. Fifty-two of the 89 underwent tonsillectomies. Clinical records of 43 of the 52 (83%) were complete and formed the study population. The ages of the study group, comprised of 25 men and 18 women, ranged from 4 to 67 years (mean: 24 years).

Clinical records were evaluated for evidence of purulence in the peritonsillar space and tonsillar abscess formation. Parenchymal tonsillar abscess was defined by identifying focal areas of neutrophils and necrotic debris within the parenchyma of the tonsil. Diagnosis of crypt abscess was made when similar findings were present within the walls of a tonsillar crypt. Purulence was present within the peritonsillar space in all 43 cases reviewed.

CASE REPORTS

Patient 1

A 37-year-old woman presented with severe right otalgia and odynophagia, unable to swallow saliva. She had been seen 2 days prior with similar complaints and was prescribed penicillin 250 mg four times daily. Swelling of the right tonsil toward the midline and tender right-sided cervical adenopathy were noted at the time of admission. The patient denied a history of recurrent pharyngitis. A 0.5-cm abscess was identified within the left tonsil.

Patient 2

A 45-year-old man presented with intolerance of liquids or solids and a 1-week history of a sore throat. He had no history of pharyngitis. Bilateral tender cervical adenopathy, trismus, deviation of the uvula, and muffled voice were noted on admission. High-dose parental penicillin was used for 72 hours without success. Microscopic examination identified reactive follicular hyperplasia with multiple microabscesses throughout the parenchyma of the left tonsil.

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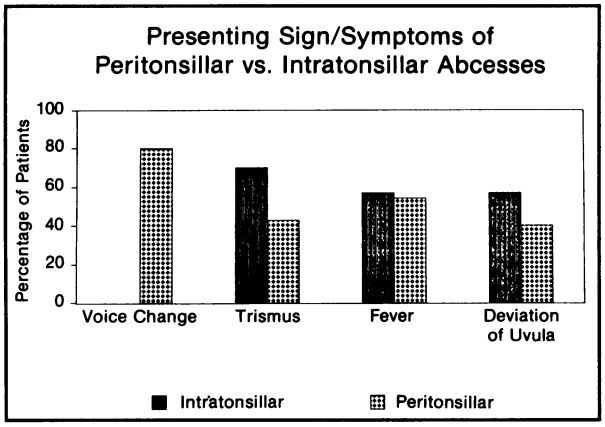

Figure 1. A comparison of the presenting signs and symptoms of peritonsillar abscess patients with and without tonsillar abscess.

Patient 3

A 16-year-old girl was treated by the emergency department with 1.2 million units of parental penicillin and was prescribed 500 mg of penicillin four times daily for a sore throat. Because of progressively increasing symptoms, she returned to the otolaryngology clinic 2 days later. There was no prior history of pharyngitis. Large, inflamed tonsils with marked exudate and bilateral cervical adenopathy were present. The left tonsil was approaching the midline. The patient received high-dose parental penicillin for 72 hours without improvement. Heterophil testing for exposure to the Epstein-Barr virus was positive. Microscopic examination of the tonsils identified focal areas of necrosis within the substance of the left tonsil.

Patient 4

A 21-year-old woman presented following 3 days of progressive increase of fever, sore throat, and right otalgia. Deviation of the uvula and tonsillar enlargement with exudate as well as bilateral tender cervical adenopathy were found. The patient had a history of recurrent pharyngitis. For 48 hours prior to tonsillectomy, high-dose parental penicillin was used without success. Pathologic examination of the operative specimens demonstrated an abscess within the tonsillar parenchyma of the right tonsil.

Patient 5

A 27-year-old woman was seen in the emergency department with complaints of sore throat, odynophagia, and trismus. She had a significant history of recurrent tonsillitis. Marked edema and erythema of the right tonsillar region with deviation of the uvula toward the left and bilateral nodes were identified. The patient received parental penicillin for 12 hours prior to tonsillectomy. Microscopic examination revealed focal areas of necrosis within the right tonsil.

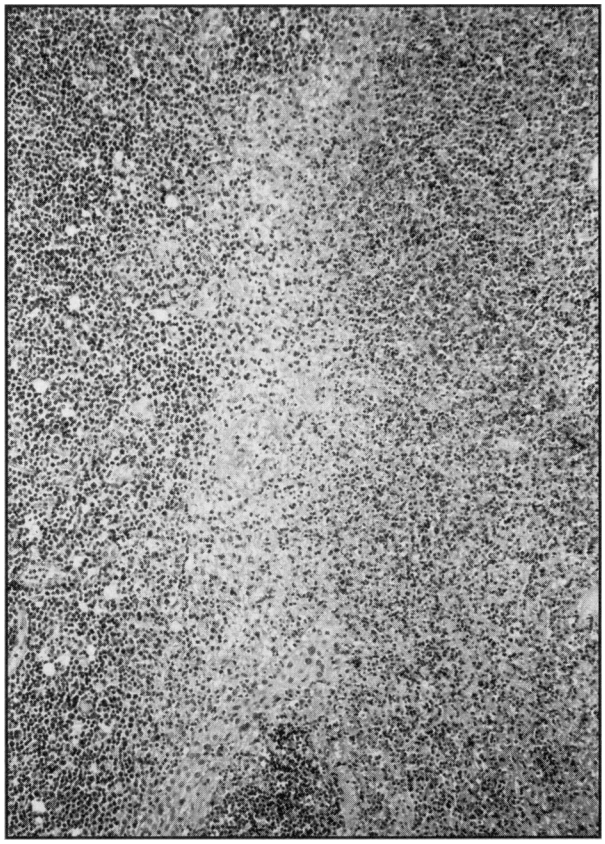

Figure 2. Photomicrograph illustrating a crypt abscess with extension into the tonsillar parenchyma (×100).

Patient 6

A 16-year-old boy presented with complaints of sore throat and neck swelling. He had experienced problems with recurrent sore throat. His pharynx was markedly erythematous but without tonsillar exudate. Bialteral cervical adenopathy was noted. After failing to respond to parental penicillin, tonsillectomy was performed 77 hours later. Pathological examination revealed four areas of microabscess formation within the right tonsil.

Patient 7

A 24-year-old man presented with a 3- to 4-week history of increasing sore throat, dysphagia, trismus, and fever to 101°F. He had a history of recurrent

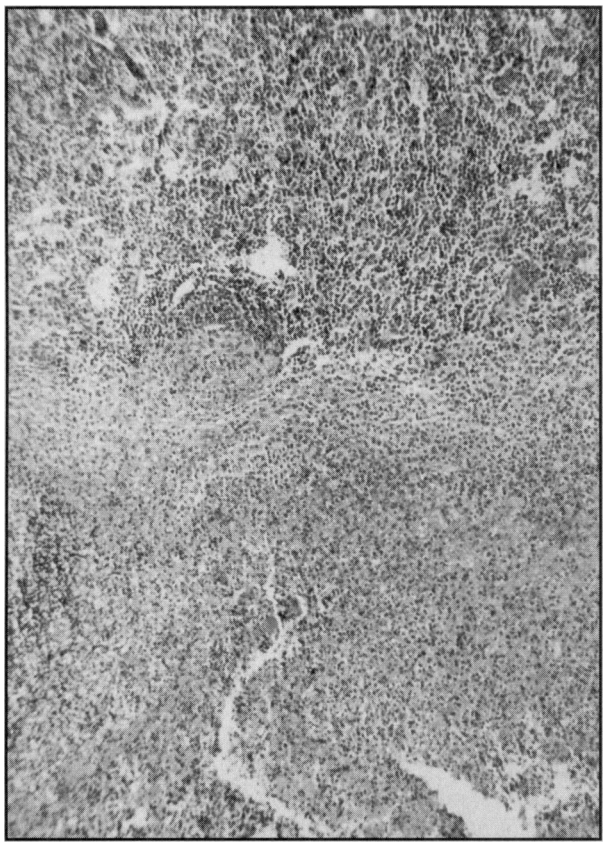

Figure 3. Characteristic parenchymal tonsillar abscess demonstrating the necrosis and inflammatory cells (×40).

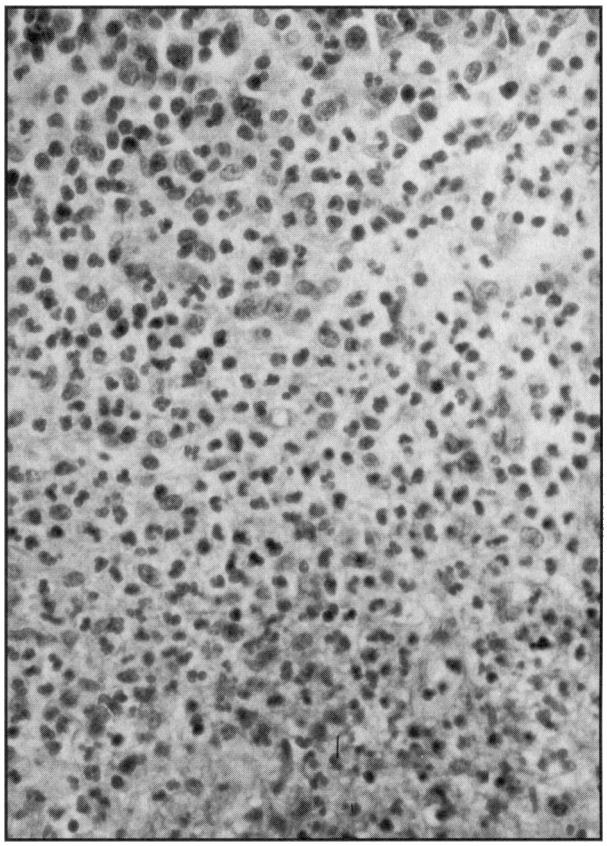

Figure 4. Higher power view of the wall of the tonsillar abscess wall (×400).

pharyngitis. On physical examination, erythema of the pharynx was noted with bilateral tender cervical adenopathy. The uvula was deviated with marked swelling of the tonsils. Pathological examination identified a crypt abscess within the right tonsil.

RESULTS

Seven (16%) of the 43 patients who underwent tonsillectomy au chaud had evidence of tonsillar abscess formation. These seven cases represent 5% of the 149 patients between 1982 and 1987 at Scott and White who were diagnosed with peritonsillar abscess. The tonsillar abscess was always identified ipsilateral to the peritonsillar abscess.

Figure 1 charts the presenting signs and symptoms of the 5 parenchymal, 2 crypt, and remaining 36 peritonsillar abscesses. The presence of cervical adenopathy and tenderness, respiratory difficulty, otalgia, tonsillar erythema with exudate, odynophagia, fever, and uvula deviation did not differ regardless of the presence or absence of a tonsillar abscess. Voice changes were not identified in any of the patients in the tonsillar abscess group, whereas 80% of patients with peritonsillar abscesses alone were symptomatic. Trismus was identi-

TABLE. INTRAOPERATIVE CULTURES

Gram-Negative	Gram-Positive
Aerobes	
Enterobacter cloacaecoaceticus	Beta *Streptococcus* group F
Klebsiella oxytoca	*Streptococcus viridans*
Pseudomonas putida	
Corynebacterium sp	
Acinetobacter calcoaceticus	Beta *Streptococcus* not group A
Eikenella corrodens	
Beta *Streptoccocus* group A	
Neisseria sp	
Anaerobes	
Bacteroides sp	*Propionibactrium* acnes
Villonella purvula	

fied almost twice as frequently (70%) in the tonsillar abscesses group compared to only 43% in the peritonsillar group.

High-dose parental penicillin was the most commonly prescribed antibiotic. The mean duration of parental antibiotic treatment prior to tonsillectomy was 44.9 hours among the seven patients with tonsillar abscess and 68.7 hours in patients without tonsillar abscesses. Eleven aerobes (six gram-negative and five gram-positive) and three anaerobes (two gram-negative and one gram-positive) were identified (Table). No pure cultures were isolated. Mixed anaerobic and aerobic organisms were identified as frequently as aerobic organisms in intraoperative cultures.

DISCUSSION

The palatine tonsils are located in the lateral portion of the oropharynx. The palatoglossal muscle defines the tonsillar sinus anteriorly and the palatopharyngeal muscle, posteriorily. Laterally, the tonsil is covered by a fibrous sheath of connective tissue. The visible medial portion and the tonsillar crypts are covered by nonkeratinized squamous epithelium. The tonsillar crypts vary in number from eight to 20 and arborize throughout the substance of the tonsil. Breaks in the crypt epithelium allow interaction of antigenetic stimuli such as bacteria and viruses with tonsillar lymphocytes.[1] Contents of the crypts are expelled by contraction of the tonsillopharyngeus muscle. Activation of the superior pharyngeal constrictors provides the stimuli for contraction of the tonsillopharyngeus.[3] Failure to clear debris from within the crypts because of structural problems[4] or local conditions may lead to inflammation and infection within the tonsil.

Acute follicular tonsillitis occurs when a virulent organism gains entrance into the tonsillar crypts and begins to replicate in the base of the crypt. Localized edema, an influx of neutrophils, and exfoliation of the crypt lining occur in response to bacterial proliferation. Clinically, these manifestations present as a red, swollen tonsil with exudate. Extension of the tonsillar infection may lead to suppuration within the peritonsillar space. Although other mechanisms may be operative,[3] direct extension occurs most often.

Tonsillar abscess formation commonly develops as a sequela of acute follicular tonsillitis. Occlusion of the crypt appears to be fundamental to the formation of a crypt abscess. The tonsillar crypt may become sealed as a result of localized inflammation infectious mononucleosis, as in patient 3. Alternatively, failure of the tonsillopharyngeus muscle to clear debris or food may provide the conditions necessary for crypt abscess formation, as in patients 6 and 7.

Parenchymal tonsillar abscesses may occur through at least two different mechanisms: direct extension of a crypt abscess into the tonsillar parenchyma as in patient 7 (Figure 2) or seeding of bacteria throughout the tonsil via the bloodstream or lymphatics. The latter appears to have been the mechanism in patients 1, 2, 4, and 5 (Figures 3 and 4). Multiple scattered areas of necrosis are best accounted for with this mechanism. Parenchymal cellulitis would be expected regardless of the mechanism. The degree of tonsillar sepsis or yet undefined factors may determine the frequency and size of parenchymal abscess formation.

The association of tonsillar abscess with peritonsillar abscess is not unexpected. Presence of peritonsillar abscess and its subsequent treatment permitted detection of the relatively transient condition of tonsillar abscess. The usual duration of acute follicular tonsillitis and peritonsillar abscess is sufficient to permit abscess formation within the tonsil. The proliferative capabilities of tonsillar lymphocytes and excellent vascular supply of the tonsil permit a rapid turnover of the necrotic debris and resolution of the abscess within 7 to 14 days, the usual time course of peritonsillar abscess. Antibiotic therapy, if instituted after tonsillar abscess formation, would be expected to shorten the time required for abscess resolution. Hence, it would not be noted on pathologic examination as frequently as it occurs. Prospective studies would be required to identify the true frequency of tonsillar abscess formation.

Intratonsillar abscesses are usually found in association with peritonsillar abscess. Absence of a distinct clinical symptom complex precludes any therapy directed toward the tonsillar abscess. Hence, therapy should be directed toward the peritonsillar abscess. Antibiotics directed against the usual bacterial agents of peritonsillar abscess would be expected to be effective therapy for the infection within the tonsil. Surgical treatment of peritonsillar abscess is adequate treatment for an intratonsillar abscess if present.

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